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Original Article

Sixth nerve palsy + ipsilateral Horner's Syndrome = Parkinson's Syndrome

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Abstract

Purpose: To present five patients with VIth nerve palsy and ipsilateral Horner's Syndrome (HS), as a result of cavernous sinus alteration.
Study design: Consecutive case series.
Material and methods: Five patients presented abducens palsy with horizontal diplopia (3 in primary position and 2 in lateral gaze only) and ipsilateral HS.
Apraclonidine 0.5% drops evidenced sympathetic denervation in all patients 40–60 min after instillation. All 5 cases had neuroimages (MRI in 3 cases, Computerized Tomography – CT in one case and Magnetic Resonance Angiography – MRA in one case) demonstrating cavernous sinus lesions; 2 meningiomas, 1 carotid-cavernous aneurism, 1 foreign body (bullet) and 1 squamous cell carcinoma.
Conclusion: Lesions on the cavernous sinus need to be considered in cases of abducens nerve palsy and ipsilateral Horner's Syndrome.

Keywords: Sixth nerve palsy, Horner's Syndrome, Cavernous sinus, Apraclonidine

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Introduction

Disfunction of the sixth (abducens) cranial nerve may result from lesions occurring anywhere along its pathway between the sixth nerve nucleus in the dorsal pons and the lateral rectus muscle within the orbit.

Horner Syndrome can be caused by damage to the sympathetic pathway at any location of its route from the hypothalamus to the eye.

The association of the sixth nerve palsy with ipsilateral Horner's Syndrome has a localizing value in the posterior cavernous sinus, also known as Parkinson's Syndrome.

The syndrome has been described mainly in aneurisms in and around the posterior cavernous sinus presenting acutely with variable pain or anesthesia–hypoesthesia in the side of the lesion.

We present five patients with VIth nerve palsy and ipsilateral Horner's Syndrome (HS), an association orienting diagnosis toward the cavernous sinus.

Material and methods

Consecutive case series of five patients with horizontal diplopia secondary to VIth nerve palsy and HS with ipsilateral cavernous sinus lesion has been registered with the institutional review board and followed the tenets of the Declaration of Helsinki. After clinical diagnosis all patients were asked for neuroimaging. The diagnosis of Horner

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Figure 1. (a) RE VI nerve palsy and HS. (b) After Apraclonidine 0.5% midriasis and lid retraction is observed. (c) Right CS tumor (squamous cell carcinoma).

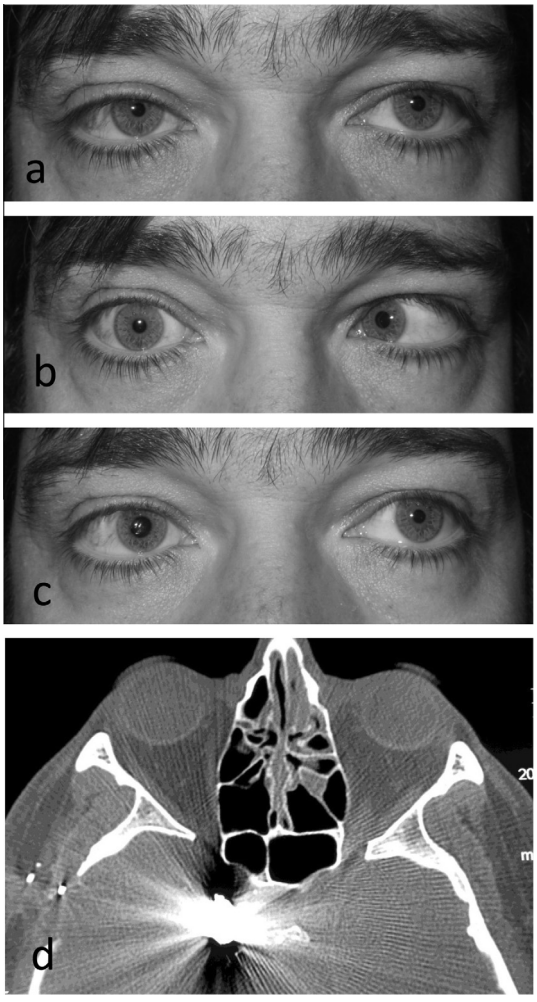

Figure 2. (a and b) RE VI nerve palsy and HS. (c) Apraclonidine 0.5% response after 40 min. (d) CT axial view shows a metallic foreign body at the right CS.

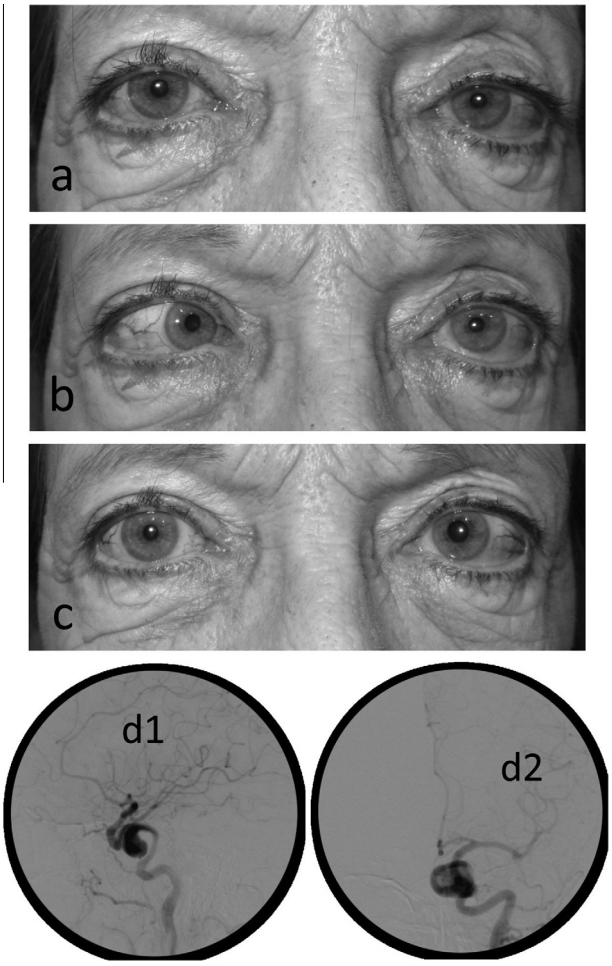

Figure 3. (a and b) Left eye VI palsy and HS. (c) Apraclonidine 0.5% response after 30 min. (d) Frozen image of carotid cine-Angiography, 1 lateral and 2 frontal view, showing Intracavernous-carotid aneurysm.

Syndrome was performed by pharmacological test with Apraclonidine 0.5% in order to confirm the sympathetic denervation feature observed (Figs. 1–4).

Results

Five patients are presented, with age range between 25 and 70 years, all patients had abducens palsy with horizontal diplopia in primary position (3 patients) or evident in lateral gaze only (2 patients) and ipsilateral Horner's Syndrome (Table 1). Apraclonidine 0.5% drops, evidenced sympathetic denervation in all patients 40–60 min after instillation. Patients had neuroimages (MRI in 3 cases, Computerized Tomography – CT in one case and Magnetic Resonance Angiography – MRA in one case) demonstrating cavernous sinus lesions; 2 meningiomas, 1 carotid-cavernous aneurism, 1 foreign body (bullet) and 1 squamous cell carcinoma.

Discussion

The cavernous sinus is a structure that contains, in its lateral (meningeal) wall the oculomotor (IIIrd nerve), trochlear (IVth nerve) and the first two divisions of the trigeminal nerve. Inside the cavernous sinus, contains the abducens (VIth nerve)

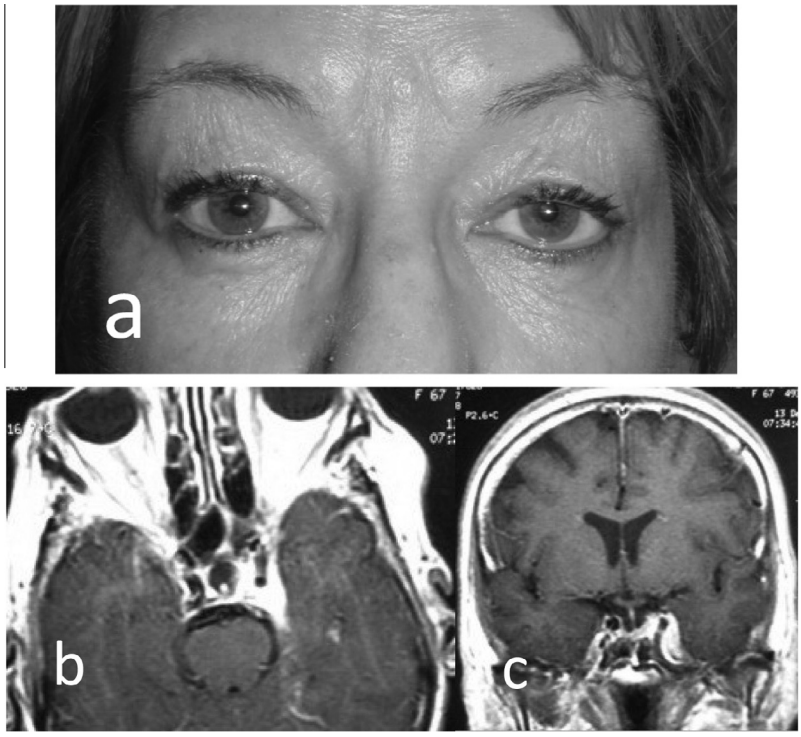

Figure 4. (a) Ptosis and miosis on LE (HS). MRI; (b) axial and (c) coronal views showing images compatible with cavernous sinus meningioma.

and the oculosympathetic nerve plexus around the internal carotid artery.

These anatomical distributions are the cause of either solitary or combined diverse impairments of cranial nerves and eventual postganglionic Horner's Syndrome.

Isolated VIth nerve palsy or Horner Syndrome alone, has no localizing value *per se*, neuroimaging or pharmacological tests are necessary to determine the injury site.

The association of VIth nerve palsy with ipsilateral HS, described by Parkinson and named after him (PS),[1,2] has a great localizing value, being the cavernous sinus the site of the lesion.

The sympathetic plexus and the VIth nerve attempt can be explained by the close anatomical relationship between these two elements inside the cavernous sinus (Fig. 5). A single lesion may produce both deficits simultaneously.

The most frequent etiologies reported for PS are; aneurism, invasive tumors, trauma, meningioma and giant cell arteritis.[3–7]

In our small series of five cases we found two meningiomas, one carotid-cavernous aneurism, 1 foreign body (bullet) and a squamous cell carcinoma as responsible for PS. Neuroimaging (MRI, CT and Angiography) confirmed the initial clinical diagnostic showing cavernous sinus lesions in all our patients.

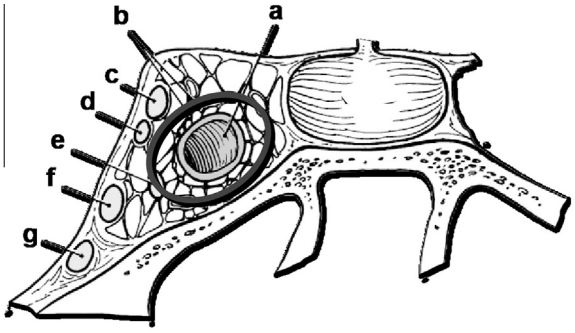

Figure 5. (a) Carotid artery, (b) sympathetic plexus, (c) III nerve (superior division), (d) IV nerve, (e) VI nerve, (f) III nerve (inferior division), (g) Maxillary nerve.

The use of Apraclonidine 0.5% has a great diagnostic value for HS but does not provide information about the level of the sympathetic pathway lesion. In our five cases helped to confirm the presence of sympathetic denervation (ipsilateral to the VI nerve deficit).[8,9]

Table 1. Clinical and ancillary findings in case series.

Patient	Diplopia	Apraclonidine 0.5%	CS imaging	Findings
WC – 32 yrs (Fig. 3)	Abd only	+	MR	Squamous cell carcinoma
CA – 27 yrs (Fig. 1)	PPG	+	CT	Metallic foreign body
JA – 67 yrs (Fig. 2)	PPG	+	MRA	CC aneurysm
MA – 58 yrs	Abd only	+	MR	CC meningioma
MC – 43 yrs	PPG	+	MR	CC meningioma

Abbreviations: PPG, primary position of gaze; abd, abduction; MR, Magnetic Resonance; CT, Computerized Tomography; MRA, Magnetic Resonance Angiography; CS, cavernous sinus; CC, carotid-cavernous.

In conclusion, neuroimaging of the cavernous sinus is recommended in the study of Parkinson's Syndrome, choosing images according to the nature of the suspected causing factor. Apraclonidine 0.5% is a helpful test for the detection of sympathetic denervation.

Conflict of interest

The authors declared that there is no conflict of interest.

References

1. Parkinson D, Johnston J, Chaudhuri A. Sympathetic connections to the fifth and sixth cranial nerves. *Anat Rec* 1978;**191**:221–6.
2. Parkinson D, Bernard, Mitchell. Horner syndrome and others? *Surg Neurol* 1979;**11**:221–3.
3. Singh M. Post traumatic Parkinson's syndrome. *Bahrain Med Bull* 2001;**23**:75–8.
4. Gutman I, Levartovsky S, Golghamer Y, et al. Sixth nerve palsy and unilateral Horner's syndrome. *Ophthalmology* 1986;**93**:913–6.
5. Jeyanth R, Pushpa J, Tripti J. *Natl Med J India* 2010;**23**:344–5.
6. Slavin JH, Guruswami A, Lally SE. A case report of unilateral Horner's syndrome and 6th nerve palsy in giant cell arteritis. In: *Poster NANOS*; 2004.
7. Kang Na Hee, Lim Key Hwan, Sung Sun Hee. Horner's syndrome with abducens nerve palsy. *Korean J Ophthalmol* 2011;**25**:459–62.
8. Morales J, Brown SM, Abdul-Rahim AS, Crosson CE. Ocular effects of apraclonidine in Horner syndrome. *Arch Ophthalmol* 2000;**118**:951–4.
9. Brown SM, Aouchiche R, Freedman KA. The utility of 0.5% apraclonidine in the diagnosis of Horner syndrome. *Arch Ophthalmol* 2003;**121**:1201–3.